MACULAR CHANGES SECONDARY TO VITREOUS TRACTION

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THE HISTORY OF A SUDDEN REDUCTION OF CENTRAL VISION and the finding of macular pathology, especially a cyst or a "hole," is often a problem in etiology.† In recent years we have been struck by the occurrence of this type of macular lesion in postoperative cataract patients who have suffered a decline in central vision after having enjoyed normal vision. The patients that we refer to have had uncomplicated surgery and postoperative courses. The integrity of the eye is unimpaired so far as we can see except that the posterior hyaloid membrane is separated from the macular region. In most instances we can see adhesions between the hyaloid membrane and the macula (Figures 1 and 2).

Our surmise is that the vitreous detachment exerts a pull on the macula through a vitreomacular adhesion. The pathology which ensues seems to be that of macular and submacular edema due to the hydraulic effect of the traction, or more probably, to ischemia because the macula is separated from its choroidal blood supply. After the elapse of weeks to months, cystic changes may appear in the foveal and perifoveal area. Abetted by the cystic changes, the traction of the hyaloid on the inner surface of the macula may pull the inner layers away giving rise to a true macular hole (Maumenee[1]) (Figure 3).

Ophthalmoscopically, the macular area appears gray, especially around the fovea where the retina is thickest. The very thin fovea is red by contrast and may be sharply demarcated because of the abrupt decrease in the ganglion cell layer at the clivus or an actual cyst or even a hole (Figure 4). The cystic changes may be seen by scatter illumination (retroillumination) as well as by biomicroscopy.

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†In referring to this macular pathology we use the term "hole" to mean the clinical appearance of a hole with the understanding that microscopically it may be a cyst, or a cherry-red spot representing the very thin fovea surrounded by the thick opaque macula, or an actual hole.

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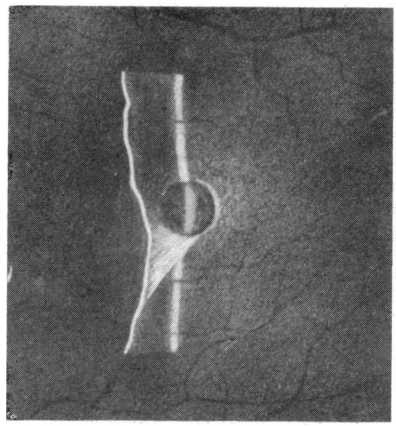


FIGURE 1

A composite drawing from several free hand sketches made at the time of fundus examinations with contact lens and slit lamp. The posterior hyaloid is shown separated from the retina, but strands from it remain attached to the macular hole.

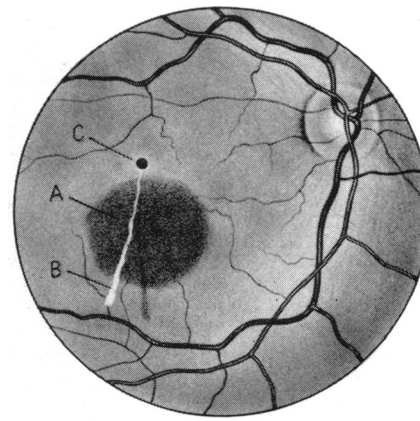


FIGURE 2

A drawing of a benign melanoma (A) with traction strand (B) extending from detached hyaloid membrane to edge of macular hole (C).

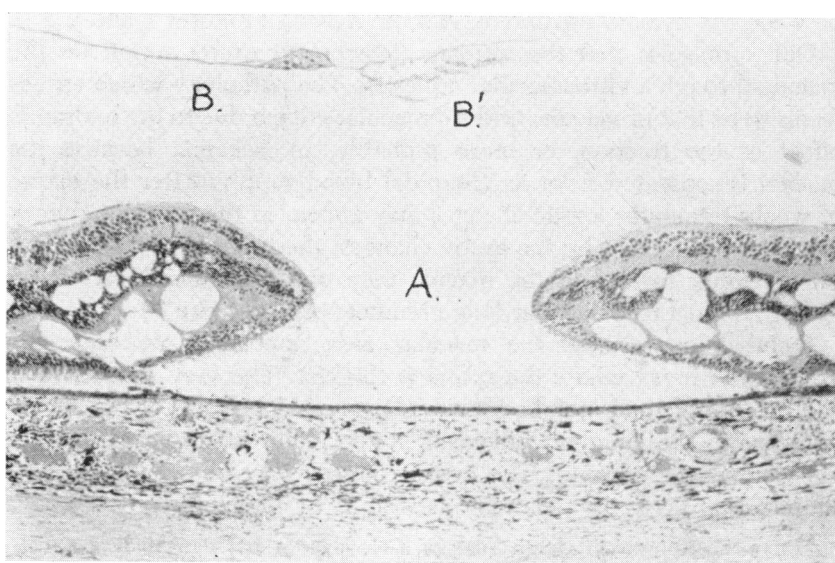


FIGURE 3

A hole (A) in the macula of an eye enucleated because of sympathetic inflammation. Abetted by the cystic changes, the traction of the hyaloid on the inner surface of the macula has pulled the inner layer away producing a true macular hole. The atrophic macular tissue adherent to the hyaloid is seen at B and B'. Section lent through the kindness of Dr. A. E. Maumenee.

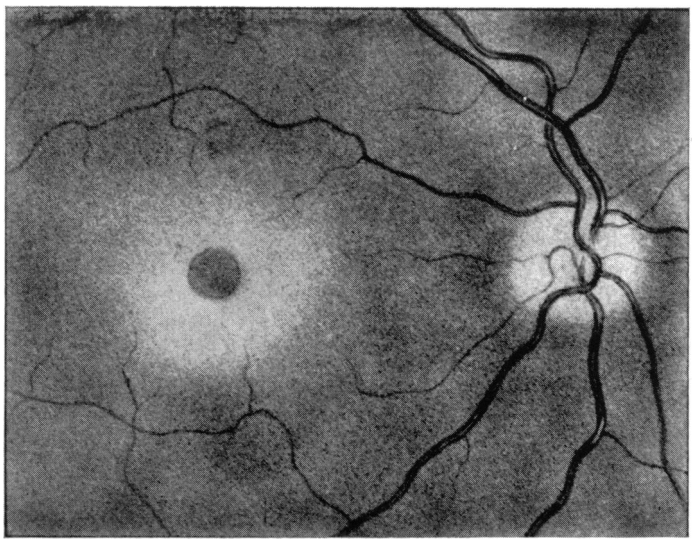


FIGURE 4. MACULAR HOLE SURROUNDED BY RETINAL CLOUDING.
A man, aged 66, gave a history of sudden reduction of vision in the left eye six years ago. This drawing was made just prior to the enucleation of this eye for an unrelated malignant melanoma of the choroid. Biomicroscopy indicated traction on the macula by the detached vitreous.

The macular pathology varies considerably in its clinical appearance. We have noted the sharply demarcated hole surrounded by the gray zone. Also, the fovea may have a poorly demarcated reddish appearance, or the macular region may show a general cloudiness or indistinctness. Schepens[2] has observed an actual break with detachment of the macula as well as hemorrhage and degenerative changes. The adhesion between the macula and the hyaloid is sufficiently weak in some cases to permit a spontaneous severance of the adhesion and restoration of central vision.

Our interest in this subject began because of an encounter with eight aphakic eyes with macular holes noted after an extraction of a senile cataract. Four of the eight had good vision from 1 to 10 years following surgery before the central vision declined. In none of these has there been an improvement during an observation period of from 1 to 6 years. Two of the eight had good postoperative vision for 3 to 6 weeks before the decline in central vision. In each case there was a return of 20/20 vision during a 3-month period. Two of the eight had poor central vision from the time of surgery, and the fundi revealed a macular hole which we felt must have preceded the surgery.

These few cases suggest that this macular pathology tends to undergo spontaneous regression with a return of normal vision if it occurs in the immediate postoperative period whereas if it occurs months to years after surgery it tends to be permanent.

Although our interest in this subject was prompted by macular changes which appear in aphakic eyes, we feel that the same macular pathology and the same mechanism prevail in phakic eyes.

The eight aphakic eyes with macular holes just discussed represent 16 per cent of the 51 macular holes we have indexed in our private files. An analysis of the remaining 43 eyes in the group of 51 yields the following:

15 eyes—The hole was thought to be due to traction on the vitreoretinal symphysis.*

6 eyes—The patients had diabetes or high blood pressure.

5 eyes—The hole was associated with benign melanomas in the macular region.

4 eyes—The hole was associated with drusen changes.

4 eyes—The hole was thought to be a feature of central angiospastic retinopathy.

2 eyes—The hole followed serous detachment of the retina.

2 eyes—The hole appeared after a commotio retinae.

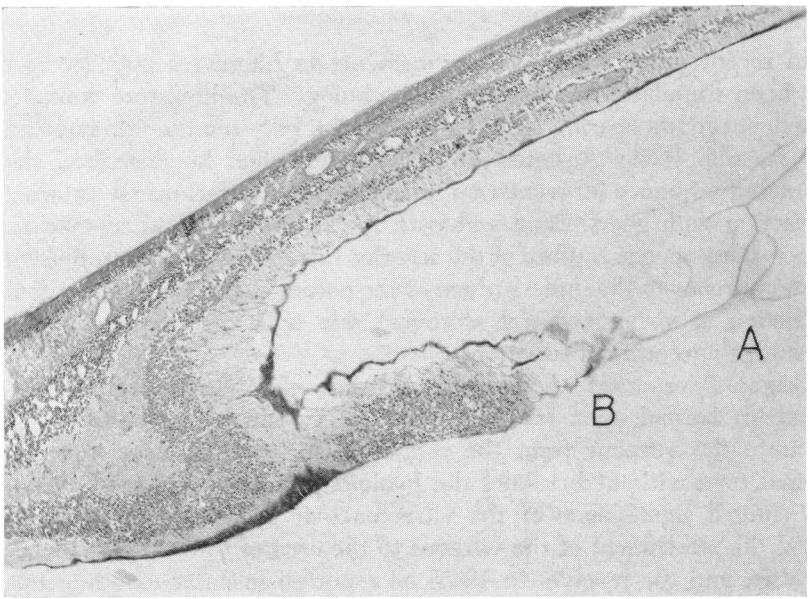
5 eyes—No associated pathology noted.

In our series the number of benign melanomas associated with a traction hole seems to be large. Most of these patients have been seen in consultation because an active melanoma was suspected. In no case have we thought this was true. We feel it is more likely that the melanoma has predisposed to developmental vitreomacular adhesions (Figure 2). It is possible that some instances interpreted as central angiospastic retinopathy and as central serous or hemorrhagic disciform detachment of the macula (Maumenee[3]) belong to the group under discussion.†

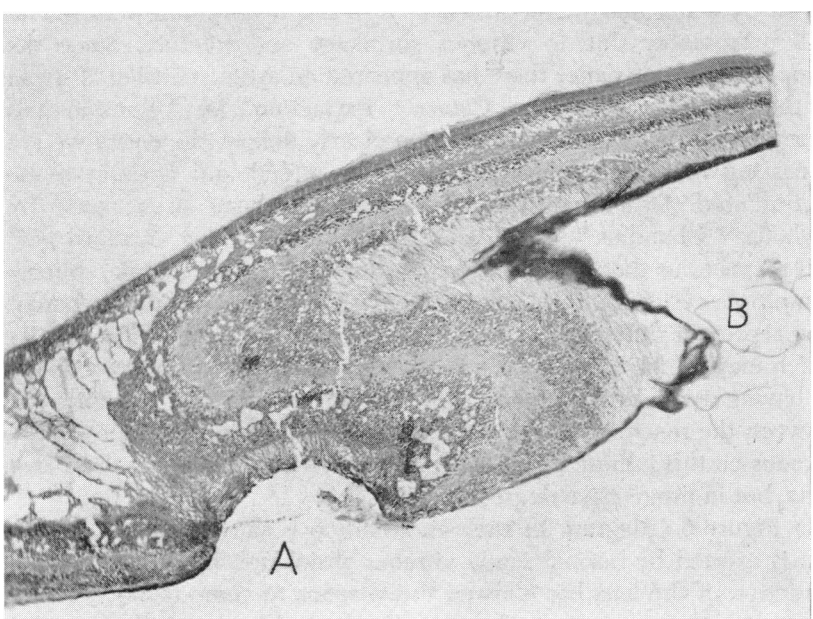
We encountered a patient who had suffered a sudden decrease in central vision in one eye 6 years prior to enucleation of the eye for malignant melanoma of the choroid. Our examination prior to the enucleation revealed a hole in the macula (Figure 4). Sections of the eye showed a retraction of the hyaloid with adhesion to the macula (Figure 5).

*We have done biomicroscopy with particular regard to detecting this lesion for only a few years and our series of macular holes covers a longer period.

†In a recent conversation with Dr. A. E. Maumenee we find that he also considers some of his cases of central serous or hemorrhagic disciform detachment of the macula as belonging to the group we are discussing.



A



B

FIGURE 5

A, a section through the macular area of the enucleated eye mentioned above. (A tongue of tissue (B) is being pulled away from the detached macula by traction from the hyaloid (A). Edema of macula and cystic degeneration of the internal nuclear layer.) B, a section through the macular area of the same eye at a different level. (The detached edematous and cystic macula with the fovea showing as a deep cup (A). There is traction at the vitreomacular symphysis (B).)

In recent years, because of refinements in biomicroscopy, interest has been stimulated in vitreoretinal pathology. The literature contains excellent reports bearing on the inter-relation between the vitreous and the macula. Irvine[4] ushered in this subject when he described the following sequence of events: an uncomplicated intracapsular cataract extraction with good vision and with the anterior hyaloid membrane intact; spontaneous rupture of the anterior hyaloid membrane; adhesion of the vitreous to the undersurface of the corneoscleral wound; gradual reduction in vision (central scotoma) due to a tug on the macula through a vitreoretinal adhesion.

Grignolo[5] reported microscopic evidence of a vitreomacular symphysis in normal eyes. He stated that it is difficult or impossible to separate the vitreous from the retina at the macular area in some normal eyes without breaking the hyaloid. Schepens has appreciated the clinical significance of the vitreomacular adhesion. In 1954[2] he noted the attachment of the vitreous to the area of the ora-pars plana, the disc, and the macula. In 1955[6] he reported macular clouding followed by a macular break which in turn led to a retinal detachment —all presumably due to vitreous shrinkage and traction. Since the completion of this paper there has appeared an article entitled "Edema of the Posterior Pole after Cataract Extraction" by Tolentino and Schepens.[7] This excellent contribution clearly defines the entity we are discussing here and it leaves little to be added and nothing to be contradicted. Other noteworthy contributions have been made by Nicholls,[8,9] Chandler,[10] Welch and Cooper,[11] and Hauer and Barkay.[12]

It seems to us that some cases of edema at the posterior pole, solitary or multiple cyst formation, hole in the macula, and macular detachment may represent different degrees or stages in a spectrum of disabilities which may all have the same mechanism. We envision the mechanism as having two essential parts. One is the presence of an adhesion between the macula and the vitreous, and the other is traction by the vitreous on this adhesion. The diagrams in Figure 6 all show these two parts, but in progressive degrees.

In Figure 6 (diagram 1) such an adhesion is shown, and mild traction is exerted by normal senile vitreous shrinkage. In Diagram 2 the extraction of the lens has allowed the vitreous to come forward and a greater traction is exerted. This may be abetted by hypotony. In Diagram 3 the addition of a rupture of the anterior hyaloid has given an added increment of traction. In Diagram 4 vitreous loss and adhesion to the wound has further strengthened the pull of the vitreous on the macula.

Now that the clinical picture and the sequence of events that pro-

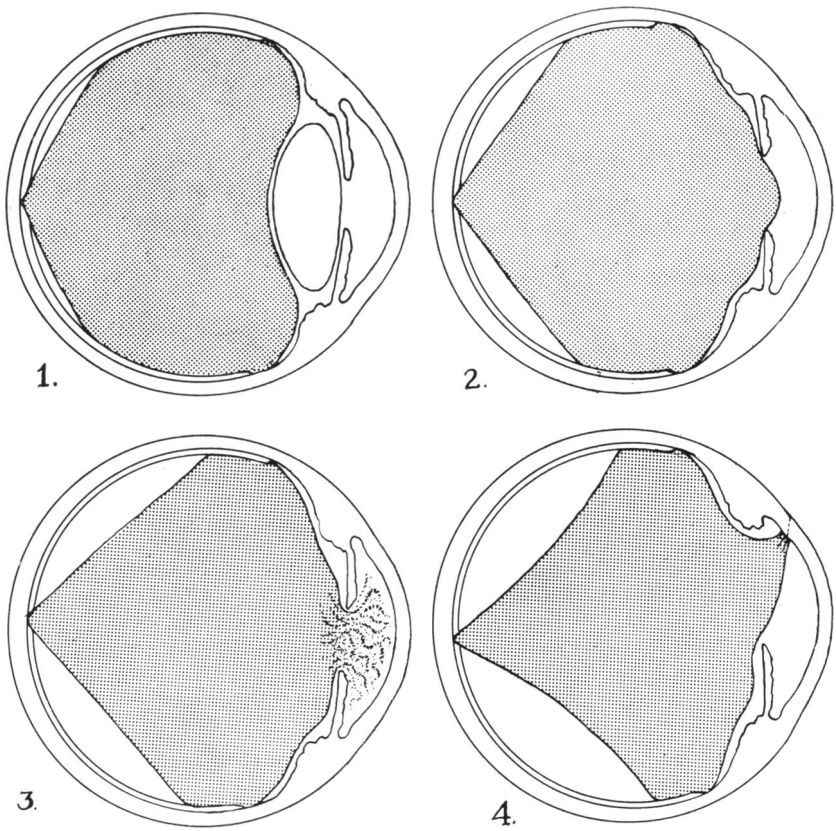


FIGURE 6

1, Vitreomacular adhesion and senile vitreous shrinkage pulling on it. 2, A similar adhesion and forward displacement of the vitreous subsequent to lens extraction. More traction is exerted on the macula. 3, The situation is similar to diagram #2 with the addition of rupture of the anterior hyaloid, vitreous movement into the anterior chamber, and consequent increased traction on the macula. 4, The same circumstances illustrated in the earlier diagrams have been potentiated by vitreous loss and attachment of the vitreous to the corneoscleral wound.

duce this macular pathology are better understood, we should give some thought to prevention and treatment. As the vitreomacular adhesion sometimes breaks spontaneously and central vision is restored, it does not seem improbable that the chain of events which lead to irreversible loss of central vision can be broken by measures designed to sever or negate the adhesions, thus restoring its blood supply to the macula. Increasing or decreasing vitreous pressure or disruption of the symphysis by ultrasonics might be worth investigation.

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DISCUSSION

DR. S. RODMAN IRVINE. First, I want to thank Dr. Reese and Dr. Jones and Dr. Cooper for sending me a copy of their paper well in advance of the meeting so that I could prepare a discussion. They have proved a supposition that I made in 1952, namely, that in certain instances vitreous may pull on the macula, in phakic and aphakic eyes, causing a central scotoma.

By careful slit-lamp examination, they have demonstrated the presence of vitreous attachments to the macula in a number of cases. In all of these cases there was collapsed vitreous, and they believe that the attachments, by virtue of the dynamics of the detached vitreous, may pull on the macula, causing edema, and cysts and holes, of varying degrees and duration, depending on the persistence of the traction effects. Macula-vitreous attachments have been assumed to be present by many authors but have been conclusively demonstrated only recently, by Goldmann, Tolentino, and Schepens, and the authors of the paper we are discussing.

The traction snubbing and rebound effects that detached vitreous exerts on the retina and the damping of such effects by the lens when it is *in situ*, as well as the exaggeration of the effects when the lens has been removed, and most especially if vitreous is in the anterior chamber (blob-like)—and attached to the corneal wound, were all discussed in detail by Anderson Hilding in two classic presentations in 1954.

The firm attachment of the vitreous at the "vitreous base" is universally

recognized, but there is great diversity of opinion as to the location and strength of other attachments, as at the lens, equator, disc, and macula, and also with regard to the types of liquefaction and collapse of the vitreous, as judged by the writings of Lindner, Grignolo, Goldmann, Pischel, Teng, and, more recently, Straatsma, Allen, and O'Malley. The only reasonable conclusion is that there is actually great variation in the secondary attachments of the vitreous and in the types of liquefaction that take place.

Clinically, we often see posterior detachment of the vitreous, in both phakic and aphakic eyes, in patients complaining of sudden vitreous opacities, lightning streaks, and transient, rapidly clearing central scotoma, and we must assume, on the basis of the work presented here that, in these cases, there has been only very temporary attachment of vitreous to the macula. After cataract extraction, the tendency for collapse of the posterior vitreous is much greater, because of the age of the patients and also because vitreous can now extend into the anterior chamber.

In my original paper on this subject, I did not prove attachment of the vitreous to the macula in those cases in which central scotoma developed some weeks or months after cataract surgery, and I assumed that the scotoma might well have been the result of iritis in some cases as the eyes with late vitreous attachments to the cataract wound were unusually irritable.

When there is actual loss of formed vitreous at operation, I believe that the central scotoma which is present in 50 per cent of these cases may be the sequela of cyclitis. The posterior vitreous always seems to be collapsed and this 50 per cent incidence is altogether too high to be attributed in every instance to vitreomacular attachments.

Because the scotoma in such cases is rather delayed and gradual in onset, Dr. Raymond Allen has offered another interesting hypothesis: that the vitreous which is adherent to the wound exerts traction on the vitreous base, wherein the retina is involved, and thus the retina is pulled directly forward, causing a tangential pull on the retina *per se*, and this pull may stretch and irritate the macula.

Of particular interest to me was the case with melanoma that developed a macular hole of unrecognized etiology and, later, sections of the eye demonstrated the relation of vitreous adhesions to the formation of the macular hole. It is possible that the primary pathology was in the retina, releasing hyaluronidase which degraded hyaluronic acid, causing loss of bound water and liquefaction of the vitreous, allowing collagen elements to coalesce and form adhesions, membranes, and tags that become firmly attached to the retina. Subsequent traction phenomena exaggerate the pathology. This was my original concept in explaining late vitreous attachments to the wound after cataract extraction and the subsequent effects on the ciliary body, vitreous base, and macula, and I believe this concept still warrants consideration. We have an example of this possibility demonstrated in lattice degeneration of the retina where the pathologic feature, as shown

by Straatsma and Allen, is liquefaction of vitreous over the degenerated area, with formation of condensation membranes and traction bands surrounding it.

In considering the whole spectrum of vitreoretinal attachments, and the alteration of these by changes in the vitreous, it is gratifying to have such evidence as Dr. Reese and his co-workers have presented today of one definite mechanism that can cause macular pathology.

DR. EDWARD W. D. NORTON. I hesitate to rise and discuss this paper now, because later on in the program I am giving, with Dr. Gass, a paper that will say much of what I am going to say now, but I do think it ought to be in the discussion of this paper so that people who review the literature in the future will have a chance to at least refer to our paper as a result of reading Dr. Reese's paper.

It is obvious that this is a very controversial subject, and as we go along we will find it a lot more controversial. I think one of the problems is trying to discover what we are talking about and be consistent in what we are talking about.

I think the changes that occur in the macula following lens extraction are one thing and that the changes that occur in the macula following retinal detachment surgery are something else. They are not necessarily the same. I think they give a different picture when you study them with fluorescein.

When I began studying the vitreous I thought these changes were all due to vitreous traction on the macula, and it is very easy to convince yourself of this when you examine the patient. However, since Dr. Gass came with me and we have recognized the characteristic picture seen in fluorescein studies, we have been very careful to study the vitreous and the macula with the contact lens and the slit-lamp as well as other techniques.

I am absolutely convinced that the great majority of these cases do not have vitreous traction on the macula. At first I felt that perhaps we were not examining it correctly. Everyone else sees it; why don't we? But after awhile when you examine many of these patients you can convince yourself that you are able to see the vitreous. It may have cells in it; it is just in front of the macula; it moves freely, and there is no evidence of tugging on the macula.

We see other patients in other diseases, in whom there is obvious pulling of the vitreous on the retina and macula, and yet they do not get these changes. I think it is important for us to say that we do not know how many of these patients have vitreous attached to the macula. We have to assume that the people seeing the attachments are really seeing them. I will just say that the majority of the patients we are seeing do not have vitreous attached to the macula. I do not know what the percentage is, but the great majority do not have it.

I also want to make it clear that we are not saying the vitreous does not play a role. I do not know what role it plays. We certainly were impressed

with the number of cells in the vitreous and the reaction in the vitreous, and we know its relationship following lens extraction when there are obvious changes in the vitreous. We think there is some relationship, but as best we can tell on our examination it is not a result of vitreous traction on the macula.

DR. BERNARD BECKER. I feel it is important to point out that there is at least one other possible contributing factor in the macular pathology following cataract surgery. I am referring to the role of epinephrine in such pathology. Epinephrine can produce a reversible disease process in the macula with loss of central vision and it is important to emphasize this fact both as a warning to clinicians and because it may provide some indication as to pathogenic mechanisms.

We have reviewed 200 consecutive patients with glaucoma treated with topical epinephrine and found maculopathy in 4 per cent of treated eyes. After more careful review, it appeared exclusively in aphakic eyes and the incidence was close to 20 per cent.

The visual losses varied from the 20/40 level to the 20/100 level. In every case it was reversible, and vision returned slowly to the original level, usually 20/20. In one instance it was 10/400, coming back to 20/50, which was the patient's original level. In many cases these patients received epinephrine before they were aphakic without any impairment of visual function, and only after they were aphakic and their glaucoma made it necessary to place them back on epinephrine did they develop central visual loss.

In three instances we have been able to reproduce the maculopathy by re-introducing epinephrine. We were able to lower the patient's vision from the 20/20 to 20/50 level, at which time we stopped therapy and permitted the patient to recover. In one patient we have done this on two occasions. In all instances the visual changes were reversible.

I point out the epinephrine maculopathy because there may be some patients in Dr. Reese's series or Dr. Norton's series who are receiving epinephrine without this being taken into account in their over-all evaluation.

DR. JOSEPH A. C. WADSWORTH. In the normal young eye the vitreous fills the vitreous cavity and offers no traction, but we know that in the senescent changes the vitreous gradually retracts and separates itself from the macula, an area that is normally closely adherent. As time goes on this normal adhesion is probably weakened.

Another rather constant finding is that after intraocular surgery a very high percentage of patients show a detachment of the vitreous. These cases probably are not in a state where these adhesions have been weakened, and we would expect to get a greater degree of traction here. One thing I am rather interested in is the fact that we do not often see macular edema after glaucoma surgery, and I would like to ask Dr. Reese if he has seen this.

DR. IRA S. JONES. We thank very much Drs. Irvine, Norton, Becker, and Wadsworth for their pertinent and interesting discussions. Dr. Irvine mentioned the diagrams which will be included in the paper, and perhaps we might show those briefly.

[Slide] In an eye in which the lens is still present, vitreous shrinkage with an attachment at the macula, if such exists, may cause some pull on the macula, and if this mechanism is an effective means (as we suggest in our paper) for causing change in macular function, a certain number of these patients may get it, if it proves that the traction of the vitreous is the trigger mechanism or is of sufficient moment.

[Slide] When the lens has been removed and the anterior hyaloid bulges through the pupil and the vitreous comes forward, it seems mechanically sound to expect a greater traction on the macula. These are the patients in whom we have found a larger number of cysts or holes.

[Slide] If the vitreous is semi-fluid at the time of surgery or if the anterior hyaloid ruptures perhaps at a later time so that the entire chamber fills with vitreous, then an even greater traction may be exerted, and one would expect a greater number of these patients to show alteration in macular function if our mechanism and our supposition is correct. We do not yet have enough data to make a definite statement.

[Slide] I will not comment on late attachments of the vitreous to the wound, but if one supposes that vitreous is lost at the time of surgery and that the remaining vitreous either is smaller in volume or attaches to the inner part of the wound or both, then an even greater traction on the macula ensues and these would be most likely to have alteration in macular function.

We do not claim—in fact, we are convinced of precisely what the discussers have suggested, that is, that macular disease may be present without vitreous traction. Vitreous traction may be present without macular disease, but we feel that when vitreous traction is present the likelihood of macular malfunction is increased.